U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 27 March 2006

CASE NO.: 2004-BLA-5868

In the Matter of:

ROBERT G. HALL,
Claimant,

v.

RANGER FUEL CORP.,
Employer,

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party-in-Interest

DECISION ON MOTION FOR RECONSIDERATION

This proceeding arises from a claimant's subsequent claim after a denial of his prior claim for benefits under the Black Lung Benefits Act, 30 U.S.C. § 901 *et seq*. (the Act) and implementing regulations, 20 C.F.R. Parts 410, 718, and 725 (Regulations). (DX-1). I issued a Decision and Order – Denying Benefits in this claim on October 31, 2005. Claimant, by counsel, submitted a Motion for Reconsideration on November 30, 2005. In his Motion, Claimant requested that I reconsider whether Dr. Rasmussen's opinion establishing legal pneumoconiosis is more consistent with the Department of Labor's (DOL) analysis of prevailing medical science and the facts of this case. Employer filed a Response to Claimant's Request for Reconsideration on March 10, 2006 at my request.

Pursuant to 20 C.F.R. § 725.479(b), any party may request reconsideration of an administrative law judge's (ALJ's) decision and order within thirty days of the date the decision and order is filed. Having timely filed his Motion for Reconsideration, the Claimant's Motion is hereby GRANTED. My analysis of Dr. Rasmussen's opinion follows.

Upon thorough and extensive review, I find that Dr. Rasmussen's opinion could be more consistent with the DOL's analysis of prevailing medical science. However, ultimately, his conclusions are unsupported by objective medical evidence.

Although I find that Dr. Rasmussen's opinion could be more consistent with prevailing medical science, further explanation is required. Specifically, I address Dr. Rasmussen's

statement "Because of the known effects of coal mine dust exposure, one **must** include coal mine dust exposure as one of the causes of Mr. Hall's lung disease." (my emphasis). I understand Dr. Rasmussen to mean that because of the clinical evidence and the known effects, one must include coal mine dust exposure as a cause in this particular case. To the extent that Dr. Rasmussen means that one must always include coal mine dust exposure as a cause in every miner's pulmonary impairment because if its known effects, I disagree and find that this position would be contrary to the Department of Labor's requirement that that a physician base his findings of pneumoconiosis on objective medical evidence. *See* 20 C.F.R. § 718.202(a)(4).

I find that Dr. Rasmussen's report as it addresses the progressive and latent nature and the additive effect that coal mine dust exposure can have is consistent with the DOL's analysis of prevailing medical science. Dr. Rasmussen cited several epidemiological studies that concluded that coal mine dust has an additive effect. Many of these studies were referenced in the response to the comments during the notice and comment stage prior to the implementation of the 2001 Amendment to the Federal Regulations as supporting evidence that coal mine dust exposure can cause pulmonary impairment in the absence of positive x-ray evidence. *See* 65 Fed. Reg. 79937-45. (December 20, 2000). The studies were referenced in response to comments made regarding the proposed amended definition of pneumoconiosis to include "legal pneumoconiosis" at 20 C.F.R. § 718.201.

Included in these citations is one to David Coggen and Anthony Newman Taylor, "Coal Mining and Chronic Obstructive Pulmonary Disease: A Review of the Evidence," Thorax, 1998, Vol. 53. The DOL cited, "[t]he combined effects of coal mine dust and smoking on FEV_1 appear to be additive." 65 Fed. Reg. 79941. The DOL concluded, "[Coggen's] study supports the Department's position." *Id*. The DOL further referenced other studies which support the position that smoking and exposure to coal mine dust are additive. *See generally,* 65 Fed. Reg. 79937-45. (December 20, 2000).

Claimant argues that the ultimate question in this matter is whether Miner's chronic obstructive pulmonary disorder and impairment in gas exchange was significantly related to or substantially aggravated by Miner's years of coal mine dust exposure. Dr. Rasmussen concluded that coal mine dust exposure is a contributing cause to Miner's pulmonary impairment. However, as stated above, Dr. Rasmussen's opinion is consistent provided that his "Because of the known effects" statement and his citations to epidemiological studies are supported by the clinical evidence.

Dr. Rasmussen's analysis asserted that Miner's impairment would improve after cessation of smoking because lung function begins to return to a level consistent with the effects of aging shortly after cessation of smoking. Dr. Rasmussen's strongest evidence to support the effect that coal mine dust exposure was having on Miner was his assertion that Miner's impairment had progressively worsened since Miner stopped smoking which would be consistent with the DOL's position that pneumoconiosis can be progressive and latent. Therefore, according to Dr. Rasmussen, because it had worsened since cessation of smoking, this was clinical evidence that there was a progressive deterioration caused from the effect of coal mine dust exposure on Miner's lung disease.

However, upon a review of the test results in this matter, I do not find there to be evidence of a progressive impairment. While this case is a subsequent claim and only the newly submitted medical evidence is reviewed in making my determination of whether Claimant has established a change in circumstances, I find it necessary to review Claimant's prior pulmonary function tests and blood gas studies to determine whether Dr. Rasmussen's assertion of a progressive impairment is correct. These test results are as set forth in the charts below.

Pulmonary Function Test (PFT)

- Post-bronchodilator value
-

Date	**Height**	**Age**	**FEV_1**	**FVC**	**MVV**	**FEV_1/FVC**	**Qualifying?**
8/12/87	70.75"	54	1.97	3.28	61	50%	Yes
9/17/87	72"	54	2.05 2.43*	3.10 3.59*	63 65*	66% 69%*	Yes Yes
9/7/88	70"	55	2.16 2.26*	3.74 3.61*	67 76*	58% 63%	Yes Yes
10/21/92	70"	59	1.93 2.10*	3.57 3.94*	67 71*	54% 53%*	Yes Yes
9/3/97	70"	64	1.22	2.38	23	51%	Yes
10/27/97	70"	64	1.29	2.59	35	49%	Yes
2/11/00	70"	66	1.96	3.96	40	42%	Yes
6/11/01	70"	67	1.54 1.67*	3.48 3.99*	45 42*	44% 42%*	Yes Yes
10/02/02	68.5"	69	.91	2.29	----	40%	Yes
11/26/03	70"	70	1.29 1.47*	3.24 3.59*	---- ----	40% 41%*	Yes Yes
3/1/04	69"	70	1.69 1.77*	3.44 3.35*	49 ----	49% 53%*	Yes Yes

Blood Gas Studies (BGS)

- Post-exercise result
-

Date	PCO2	PO2	Qualifying?
8/12/87	38 34*	77 71*	No No
9/7/88	39	79	No
10/21/92	37 37*	70 71*	No No
9/3/97	35 35.7*	71 61*	No Yes
2/11/00	36 34*	62 60*	Yes Yes
6/11/01	32 34*	80 64*	No Yes
10/1/02	39.3 36.8*	63.2 87.8*	No No
11/26/03	35 36*	66 57*	No Yes
3/1/04	40	69	No

All PFT results are qualifying. However, the pattern over the period of 1987 to 2004 shows marked variability. Miner's FEV_1 result in 1987 was 1.97, in 2004 it was 1.69. These numbers have ranged from .99 to 2.16 (pre-bronchodilator) and 1.47 to 2.43 (post bronchodilator). Miner's 2004 results are improved over his 2003 and 2002 results.

Miner's blood gas study results also vary in range. Most of Miner's BGS results do not qualify. Miner had one qualifying resting result and four qualifying post-exercise results. Of the four post-exercise results, the PO_2 results vary from 61 in 1997, 60 in 2000, 64 in 2001, and 57 in 2003.

Even if I were to ignore the 2002 BGS results as argued by Claimant, the medical evidence does not show a pattern of progressive impairment – there is considerable variability. Thus, I find that Dr. Rasmussen's assertion of a progressive impairment is contrary to the medical evidence in this case.

Because the progressive impairment was fundamental to my reading of Dr. Rasmussen's conclusion, I find that his conclusion is unsupported except for his generalized statement of "Because of the known effects." Ultimately, Dr. Rasmussen's conclusion is based on the premise that because studies show that coal mine dust has an additive effect then this is conclusive evidence that it is a contributing cause in this case. However, a medical opinion based upon generalities, rather than specifically focusing upon the miner's condition, may be rejected. *Knizer v.Bethlehem Mines Corp.*, 8 B.L.R. 1-5 (1985). As I stated above, this absolute

requirement to include coal mine dust as a cause of a miner's pulmonary impairment would be contrary to the DOL's requirement that findings be based on objective medical evidence.

I find that Dr. Crissali and Dr. Zaldivar are not entirely consistent with the position of prevailing medical science, but their opinions are not hostile to the Act. While it would not necessarily be contrary to prevailing medical opinion that a miner's pulmonary impairment is entirely caused by smoking, particularly if one agrees that cigarette smoke is a more potent agent in producing emphysema than coal workers' pneumoconiosis as asserted by Dr. Zaldivar (EX-5 at 20), I find that their opinions do not give proper consideration to Miner's 11.54 years in the mines. Furthermore, I find that their reports do not give adequate consideration to the effects that coal mine dust exposure is found to have on miners as it relates to the facts of this particular case, particularly when there is a negative x-ray and an alternative cause, namely smoking.

Both Dr. Crisalli and Dr. Zaldivar concluded that Miner's pulmonary impairment was entirely caused by Miner's smoking history. Neither Dr. Crisalli nor Dr. Zaldivar adequately explained why coal mine dust did not exacerbate Miner's emphysema, which was caused by cigarette smoking.

In particular, Dr. Zaldivar listed the bases for his conclusions in his report. Dr. Zaldivar stated, "coal mine dust exposure did not contribute to obstructive impairment because 1. Miner didn't work in the coal mines for a long period of time, 2. Miner wasn't always necessarily in an area that was dusty, 3. Chest x-ray doesn't show retention of dust." *Id.* at 21-2.

First, although shorter in time that Miner's smoking history, 11.54 years of coal mine dust exposure is significant and enough to cause pneumoconiosis, and I do not find that Dr. Zaldivar gave adequate consideration to this fact. Second, the fact that Miner was a general laborer and worked in various locations does not support a conclusion of "Miner wasn't always necessarily in an area that was dusty." This is speculation on Dr. Zaldivar's part unless Miner provided this information which is not apparent from his report. Finally, Dr. Zaldivar repeatedly states that the chest x-ray "doesn't show retention of dust." However, Dr. Zaldivar explained in his deposition that chest x-rays show an inflammatory response to dust that is retained. *Id.* at 22. I fail to see how Dr. Zaldivar arrived at the conclusion that the lack of radiographical evidence of inflammation became direct evidence that little to no dust was retained, particularly when he acknowledged that a pathologist can still find macules of dust despite a negative chest x-ray. *Id.* at 29.

Also, Dr. Zaldivar, in his deposition testimony, stated that in order to attribute emphysema to dust exposure, one would expect to see radiographic evidence that a significant amount of dust was retained. *Id* at 22. My understanding of Dr. Zaldivar's position is that he believes that in order to have legal pneumoconiosis in the form of emphysema, there almost always is a positive chest x-ray. Yet, legal pneumoconiosis encompasses disease of the lungs beyond that of clinical pneumoconiosis and the existence of legal pneumoconiosis is not dependent on a finding of clinical pneumoconiosis. Dr. Zaldivar acknowledged that coal mine dust can cause emphysema but that it would be centriacinar emphysema and Miner had panacinar emphysema which manifests in bulla. *Id.* at 27. However, later, Dr. Zaldivar acknowledges that Miner should have centriacinar emphysema but that it would require a biopsy to diagnose it, and he acknowledged that coal mine dust can cause the full spectrum of

emphysema including, apparently in cases of complicated pneumoconiosis, bulla. *Id.* at 27-8, 52. Finally, Dr. Zaldivar acknowledged that coal mine dust exposure can cause a measurable impairment in pulmonary function but asserted that rarely would it cause a disabling impairment. *Id.* at 36. However, Dr. Zaldivar never addressed why coal mine dust exposure was not having an additive effect, causing even a small measurable increase in impairment, on Miner's smoking-related emphysema despite these acknowledgements.

Dr. Crisalli did state that the pulmonary function studies show variation and that this was inconsistent with coal miners' pneumoconiosis (CWP) because CWP creates a fixed obstruction. Dr. Crisalli recorded Miner's smoking history as 11 pack years and noted that his history would typically be insufficient to cause the severity of Miner's impairment. He stated, "One must wonder whether there isn't some sort of hereditary predisposition to developing emphysema as well." Dr. Crisalli stated that an 11 pack year history would be sufficient to cause this severity in a susceptible person but also stated that a 12-13 year coal mine history is significant in a susceptible person. (EX-6 at 11-12)

Dr. Crisalli concluded that coal mine dust exposure was not contributing at all to Miner's impairment. *Id.* at 37-8. However, Dr. Crisalli did not speak to the reason why a portion of that impairment could not be attributed to coal mine dust exposure. While the pulmonary function studies showed some variation, all physicians acknowledged that Miner's smoking history also contributed to the impairment. Dr. Crisalli does not address whether the variability could be attributed to the portion of the impairment that was caused by smoking. Dr. Crisalli, instead, opined of a possible hereditary predisposition than address the possible additive effect – a contribution to the level of impairment – that coal mine dust may have been causing. *Id.* at 13, (EX-2). Dr. Crisalli stated that this extensive case of emphysema is not typical of what one would see in coal workers' pneumoconiosis. *Id.* at 21. He stated that coal dust induced emphysema would not progress to the point where one sees it to such dramatic effect radiographically as in this case. *Id.* at 22.

While I am troubled by their failure to address a possible additive effect that coal mine dust exposure had on the effects of Miner's smoking history, I find that the opinions of Drs. Crisalli and Zaldivar are not hostile to the Act. Neither physician stated a position that was contrary to the spirit and purposes of the Act. Thus, although I must accord some weight to their opinions, I accord them little weight. Having accorded little weight to Dr. Dababnah and Dr. Mullins in my original decision and according little weight to Dr. Rasmussen based on the above analysis, I find that Claimant has not met his burden of showing by a preponderance of the evidence by reasoned medical opinion that he suffers from pneumoconiosis. Therefore, this analysis does not change my weighing of all the evidence under 20 C.F.R. § 718.202(a) as set forth in my Decision and Order of October 31, 2005. Claimant has failed to establish the existence of pneumoconiosis.

ORDER

My Decision and Order dated October 31, 2005, in which I denied the Black Lung benefits application of Robert G. Hall, is hereby affirmed.

A

MICHAEL P. LESNIAK
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: During the consideration of a request for reconsideration, the time for appeal to the Benefits Review Board is suspended. 20 C.F.R. § 725.479(c) (2001). Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days of the date this Decision and Order was filed in the office of the District Director, by filing a notice of appeal with the Benefits Review Board at P.O. Box 37601, Washington, DC 20013-7601. A copy of a notice of appeal must also be served on Allen Feldman, Esq. Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N-2117, 200 Constitution Avenue, NW, Washington, D.C.